We claim:

1. A method of treating a non-neoplastic hyperproliferative cell or excessive cell accumulation disorder in a patient in need thereof, said method comprising administering to said patient a therapeutically effective amount of an EphA2 agonistic agent, wherein said EphA2 agonistic agent binds EphA2 and increases EphA2 cytoplasmic tail phosphorylation, increases EphA2 autophosphorylation, increases EphA2 degradation, reduces a pathology-causing cell phenotype, or reduces EphA2 activity wherein said activity is not autophosphorylation.

2. The method of claim 1 wherein said non-neoplastic hyperproliferative cell or excessive cell accumulation disorder is a hyperproliferative epithelial cell disorder selected from the group consisting of asthma, chronic pulmonary obstructive disease, lung fibrosis, asbestosis, IPF, DIP, UIP, kidney fibrosis, liver fibrosis, other fibroses, bronchial hyper responsiveness, psoriasis, and seborrheic dermatitis.

3. The method of claim 2, wherein a pathology-causing cell phenotype of said hyperproliferative epithelial cell disorder is secretion of mucin, differentiation of an EphA2-expressing cell into a mucin-secreting cell, secretion of inflammatory factors, or epithelial or endothelial cell hyperproliferation.

4. The method of claim 1 wherein said non-neoplastic hyperproliferative cell or excessive cell accumulation disorder is a hyperproliferative endothelial cell disorder selected from the group consisting of restenosis, hyperproliferative vascular disease, Behcet's Syndrome, atherosclerosis, and macular degeneration.

5. The method of claim 1 wherein said non-neoplastic hyperproliferative cell or excessive cell accumulation disorder is a hyperproliferative fibroblast cell disorder.

6. The method of claims 4 or 5, wherein a pathology-causing cell phenotype of said hyperproliferative endothelial cell disorder is increased cell migration, cell volume, secretion of extracellular matrix molecules, secretion of matrix metalloproteinases, or endothelial cell hyperproliferation.

7. The method of claim 1 wherein said EphA2 agent is an antibody or antigen binding fragment thereof.

8. The method of claim 1 wherein said EphA2 agent is chosen from the group consisting of small molecule agonists, enzymatic activity antagonists, ribozymes, siRNA, and EphA2 antisense molecules.

9. The method of claim 7 wherein the said antibody is a monoclonal antibody.

10. The method of claim 9 wherein said monoclonal antibody binds EphA2 with a K_{off} of less than $3x10^{-3}$ s^{-1} under conditions appropriate for antibody-EphA2 binding.

11. The method of claim 9 wherein said monoclonal antibody is Eph099B-102.147, Eph099B-208.261, Eph099B-210.248, or B233 or comprises a CDR from Eph099B-102.147, Eph099B-208.261, Eph099B-210.248, or B233.

12. The method of any of claims 7, 9, or 10 wherein said monoclonal antibody is a human antibody.

13. The method of any of claims 7, 9, 10, or 11 wherein said monoclonal antibody is humanized.

14. The method of claim 1 wherein said administration increases EphA2 phosphorylation in a treated cell relative to the level of EphA2 phosphorylation in an untreated cell.

15. The method of claim 1 wherein said administration decreases EphA2 expression in a treated cell relative to the level of EphA2 expression in an untreated cell.

16. The method of claim 1 further comprising the administration of one or more additional non-neoplastic hyperproliferative cell or excessive cell accumulation disorder therapies.

17. The method of claim 16, wherein said pathology-causing epithelial or endothelial cell phenotype is secretion of mucin, differentiation of an EphA2-expressing cell into a mucin-secreting cell, secretion of fibronectin, secretion of inflammatory factors, or epithelial or endothelial cell hyperproliferation.

18. A method of treating asthma or chronic obstructive pulmonary disease in a patient in need thereof, said method comprising administering to said patient a therapeutically effective amount of one or more EphA2 agonistic agents, wherein said EphA2 agonistic agent binds EphA2 and increases EphA2 cytoplasmic tail phosphorylation, increases EphA2 autophosphorylation, increases EphA2 degradation, reduces a pathology-causing cell phenotype, or reduces EphA2 activity wherein said activity is not autophosphorylation.

19. A method of treating restenosis in a patient in need thereof, said method comprising administering to said patient a therapeutically effective amount of one or more EphA2 agents, wherein said EphA2 agent binds EphA2 and increases EphA2 cytoplasmic tail phosphorylation, increases EphA2 autophosphorylation, increases EphA2 degradation, reduces a pathology-causing cell phenotype, or reduces EphA2 activity wherein said activity is not autophosphorylation.

20. The method of claim 19, wherein said pathology-causing endothelial cell phenotype is cell migration, cell volume, secretion of extracellular matrix molecules, secretion of matrix metalloproteinases, or endothelial cell hyperproliferation.

21. The method of claim 18 or 19 wherein said EphA2 agent is an antibody or antigen binding fragment thereof.

22. The method of claim 21 wherein the said antibody is a monoclonal antibody.

23. The method of claim 22 wherein said monoclonal antibody binds EphA2 with a K_{off} of less than $3x10^{-3}$ s^{-1} under conditions appropriate for antibody-EphA2 binding.

24. The method of claim 22 wherein said monoclonal antibody is Eph099B-102.147, Eph099B-208.261, Eph099B-210.248, or B233.

25. The method of any of claims 21, 22, or 23 wherein said monoclonal antibody is a human antibody.

26. The method of any of claims 21, 22, 23, or 24 wherein said monoclonal antibody is humanized.

27. The method of any of claims 1, 15, or 17 further comprising the administration of one or more immunomodulatory agents.

28. The method of claim 27 wherein said immunomodulatory agent is an antibody that immunospecifically binds IL-9.

29. The method of any of claims 1 or 17 further comprising the administration of one or more anti-viral agents.

30. The method of claim 29 wherein said anti-viral agent is an anti-RSV agent.

31. A method of diagnosing a non-neoplastic hyperproliferative cell or excessive cell accumulation disorder or monitoring the efficacy of therapy for a non-neoplastic hyperproliferative cell or excessive cell accumulation disorder in a patient known to or suspected to have said disorder, said method comprising:

a) contacting cells of said patient with an EphA2 antibody that agonizes EphA2, decreases EphA2 activity, or decreases a pathology-causing cell phenotype; and

b) detecting EphA2 antibody binding to said cells,

wherein detecting a higher EphA2 antibody binding level than in a control patient that does not have a non-neoplastic hyperproliferative cell or excessive cell accumulation disorder indicates that the patient has a hyperproliferative cell or excessive cell accumulation disorder.

32. The method of claim 31 wherein said non-neoplastic hyperproliferative cell or excessive cell accumulation disorder is selected from the group consisting of asthma, chronic pulmonary obstructive disease, lung fibrosis, bronchial hyper responsiveness,

psoriasis, seborrheic dermatitis, cystic fibrosis, restenosis, hyperproliferative vascular disease, Behcet's Syndrome, atherosclerosis, and macular degeneration.